OXYGEN CONSUMPTION AFTER CARDIOPULMONARY BYPASS SURGERY IN CHILDREN: DETERMINANTS AND IMPLICATIONS

Jia Li, MD[a]
Ingram Schulze-Neick, MD[a]
Christopher Lincoln, MD[b]
Darryl Shore, MD,[b]
Michael Scallan, MD[c]
Andrew Bush, MD, FRCP[d]
Andrew N. Redington, MD, FRCP[a]
Daniel J. Penny, MD[a]

Objective: **We sought to assess oxygen consumption and its determinants in children shortly after undergoing cardiopulmonary bypass operations.** ***Methods:*** **Twenty children, aged 2 months to 15 years (median, 3.75 years), undergoing hypothermic cardiopulmonary bypass operations were studied during the first 4 hours after arrival in the intensive care unit. Central and peripheral temperatures were monitored. Oxygen consumption was continuously measured by using respiratory mass spectrometry. Oxygen delivery was calculated from oxygen consumption and arterial and mixed venous oxygen contents, which were sampled every 30 minutes. Oxygen extraction was derived by the ratio of oxygen consumption and oxygen delivery. Arterial blood lactate levels were measured every 30 minutes.** ***Results:*** **There was a correlation between oxygen consumption and age in patients older than 3 months (r = –0.76). Mean oxygen consumption increased by 14.7% during the study. The increase in oxygen consumption was correlated with the increase in central temperature (r = 0.73). Nine patients had an arterial lactate level above 2 mmol/L on arrival. There were no significant differences in oxygen consumption, oxygen delivery, and oxygen extraction between the group with lactate levels between 2 and 3 mmol/L and the groups with normal lactate levels both on arrival and at 2 hours. One patient with a peak lactate level of 6.8 mmol/L had initially low oxygen delivery (241.3 mL · min^{-1} · m^{-2}).** ***Conclusions:*** **During the early hours after a pediatric cardiac operation, the increase in oxygen consumption is mainly attributed to the increase in central temperature. Oxygen consumption is negatively related to age. Mild lactatemia is common and does not appear to reflect oxygen delivery or oxygen consumption or a more complicated recovery. (J Thorac Cardiovasc Surg 2000;119:525-33)**

The balance between the systemic delivery and consumption of oxygen is critical in the optimization of tissue metabolism in sick patients, including those having undergone cardiopulmonary bypass. Changes in systemic oxygen balance have been well described in the adult after cardiopulmonary bypass, including reductions in systemic oxygen delivery (Do_2) caused by diminished cardiac output and increases in systemic oxygen consumption (Vo_2),[1-3] that are related to central body temperature[1] and the systemic inflammatory response.[2] However, few data have assessed the changes that occur in systemic oxygen balance in the early hours after cardiopulmonary bypass in children.[4,5] Nonetheless, it is likely that these changes may differ from those that occur in adults for a number of reasons. First, resting Vo_2 is greater in young children than in adults.[6] Second, deep hypothermia and circulatory arrest are commonly used during a pediatric cardiac operation, which may alter Vo_2 and Do_2. Finally, the biochemical and hormonal responses to this stress have been reported to be greater after a cardiac operation in infants and children than in adults.[7,8] In the early post-

From the Cardiothoracic Unit,[a] Great Ormond Street Hospital for Children NHS Trust, London; and the Departments of Cardiac Surgery,[b] Anaesthesia,[c] and Paediatrics,[d] Royal Brompton and Harefield NHS Trust, London, United Kingdom.

Supported by the British Heart Foundation.

Received for publication July 21, 1999; revisions requested Sept 15, 1999; revisions received Oct 13, 1999; accepted for publication Oct 18, 1999.

Address for reprints: Professor Andrew N. Redington, Professor of Cardiology, Cardiothoracic Unit, Great Ormond Street Hospital for Children NHS Trust, Great Ormond St, London WC1N 3JH (E-mail: reding@ibm.net).

0022-5223/2000 $12.00 + 0 **12/1/103731**
doi:10.1067/mtc.2000.103731

Table I. *Preoperative clinical data for the 20 patients*

Patient No.	*Sex*	*Age (y)*	*Weight (kg)*	*Body surface area (m^2)*	*Diagnosis*
1	F	6.00	14.7	0.66	MV prolapse
2	F	0.23	3.9	0.23	VSD
3	M	8.00	28.8	1.04	ASD
4	M	3.25	11.6	0.55	DORV, PAPVD
5	F	5.00	18.6	0.78	VSD with PT banding
6	M	4.00	19.1	0.80	ASD
7	M	4.10	15.4	0.64	TOF
8	F	15.00	48.3	1.46	Redo TOF
9	F	8.10	26.8	0.94	Cleft MV
10	F	3.75	12.7	0.58	Sinus venous defect
11	M	6.25	22.5	0.90	TOF
12	M	3.00	13.1	0.59	VSD RVOTO
13	M	1.33	10.4	0.47	TOF
14	M	2.00	10.6	0.47	AVSD with PT banding
15	M	0.25	4.5	0.29	VSD
16	F	0.17	3.7	0.24	AVSD
17	F	6.75	22.3	0.86	AVSD
18	F	2.25	11.9	0.54	DCRV, VSD
19	M	4.70	14.9	0.64	AVSD, VSD, ASD
20	F	0.92	6.3	0.35	AVSD

MV, Mitral valve; *VSD,* ventricular septal defect; *ASD,* atrial septal defect; *DORV,* double-outlet right ventricle; *PAPVD,* partial anomalous pulmonary venous drainage; *PT,* pulmonary trunk; *TOF,* tetralogy of Fallot; *RVOTO,* right ventricular outflow tract obstruction; *AVSD,* atrioventricular septal defect; *DCRV,* double-chambered right ventricle.

operative period, an important goal of therapy is to achieve optimal tissue oxygenation at the lowest energy cost, particularly within the injured myocardium. Although the incidence of serious tissue hypoperfusion is now low, relative tissue hypoxia because of marginal tissue perfusion, which is often accompanied by lactate acidosis, remains relatively common in patients undergoing cardiopulmonary bypass.[3,9-11] The causes, implications, and optimal management of this imbalance between DO_2 and tissue demands are less well known, however. The aim of this study was to obtain a better understanding of VO_2, together with its determinants, early after pediatric cardiac operations.

Methods

Patients. This study was approved by our local ethical committee, and written informed consent was obtained from the parents of 20 children (10 boys and 10 girls) undergoing a cardiac operation with hypothermic cardiopulmonary bypass. The patients' ages ranged from 2 months to 15 years (median, 3.75 years). The clinical characteristics of the patients are shown in Table I.

Technique of anesthesia, cardiopulmonary bypass, and intensive care treatment. All patients were premedicated with 1.5 mg/kg oral trimeprazine, 30 μg/kg atropine, and 0.5 mg/kg morphine (Triatromorph). Anesthesia was induced with the inhalational agent sevoflurane (3%-7% in a mixture of gases of 50% oxygen and 50% nitrous oxide). All children were intubated with cuffed endotracheal tubes (Mallinckrodt Medical, Northampton, United Kingdom). Anesthesia was subsequently maintained with inhaled isoflurane, intravenous fentanyl (8-15 μg/kg), and pancuronium (0.2-0.3 mg/kg).

Cardiopulmonary bypass was performed under conditions of moderate to deep hypothermia with the lowest nasopharyngeal temperature between 15°C and 33°C (median, 27°C), nonpulsatile flow, and cold crystalloid cardioplegia. A flow on cardiopulmonary bypass ranging from 100 to 150 mL · min^{-1} · kg^{-1} was maintained, and the mean perfusion pressure was adjusted between 40 and 50 mm Hg by using isoflurane to vasodilate and metaraminol to vasoconstrict when required. Cardiopulmonary bypass was maintained for 24 to 157 minutes (median, 58 minutes), and aortic crossclamping was maintained for 13 to 90 minutes (median, 38 minutes). Patients were continued on bypass for between 6 and 64 minutes (median, 27 minutes) during rewarming until the central body temperature was restored to 36.9°C ± 0.4°C (mean ± SD) and hemodynamic stability was achieved. A pulmonary arterial line was inserted before the termination of bypass. Positive-pressure ventilation was recommenced after the discontinuation of bypass.

On return to the intensive care unit, the patients' lungs were mechanically ventilated by using volume-cycled intermittent positive-pressure ventilation (Servo ventilator 900C; Siemens Medical Systems, Solna, Sweden). All received continuous intravenous infusions of vecuronium (74-256 μg · kg^{-1} · h^{-1}), morphine (40 μg · kg^{-1} · h^{-1}), and midazolam (150 μg · kg^{-1} · h^{-1}) during the study period. The minute volume was adjusted to give an arterial carbon dioxide tension of 4 to 6 kPa. The inspiratory oxygen concentration was less than 60% in all

patients. All patients were naturally rewarmed without any external heating, except for one young infant of 2 months who was nursed in a Babytherm bed (4200 Dräger; Draeger Medical Limited, Hemel Hemstead, United Kingdom) and a 15-year-old girl with a central-to-skin temperature gradient of 10°C who was covered with an external heating blanket. Dopamine, dobutamine, and glyceryl trinitrate were used according to each patient's hemodynamic condition. After the study, patients were allowed to awaken when body temperature and circulation were stable. Patients were extubated on the basis of clinical judgment. All patients underwent echocardiography, which excluded residual intracardiac shunting.

Methods of measurements

Patient monitoring. All children had continuous invasive monitoring of systemic and pulmonary arterial and central venous pressures. Heart rate was continuously monitored, and the central body temperature (rectal) and peripheral temperature on the skin at the great toe were monitored with standard temperature probes (Hewlett Packard, Bracknell, United Kingdom).

Oxygen consumption. Vo_2 was measured continuously by using on-line respiratory mass spectrometry according to our previously described method.[12] This is a highly sensitive and accurate method for continuous gas analysis that allows simultaneous measurements of multiple gas fractions within a mixture. An Amis 2000 quadrupole mass spectrometer (Innovision A/S, Odense, Denmark) was adapted for use in patients ventilated with the Servo ventilator 900C. Vo_2 was measured by using the mixed expirate inert gas (argon) dilution method.[13] This requires analysis of inspired and expired gases, together with the collection of all expired gas. Before the study, the cuff of the endotracheal tube was inflated to prevent leak. The pressure within the cuff was measured with a manometer, and in all cases, to prevent hypoperfusion of the airway mucosa, pressure was maintained below arterial diastolic pressure. We performed a 2-point calibration of the mass spectrometer, exposing the distal inlet both to a 4-gas calibration mixture (nitrogen, oxygen, carbon dioxide, and argon) and to zero gas (closed inlet). This calibration was repeated at 30-minute intervals throughout the study period. The calibration of tracer gas flow (argon) was achieved by using a designated flowmeter (CT Platon, Basingstoke, United Kingdom; accuracy, ±1.25%). Ambient humidity, temperature, and atmospheric pressure were recorded from an electronic barometer (BA-888; Oregon Scientific, Portland, Ore).

Do_2, oxygen extraction, and lactate levels. Measurements were made for the first 4 hours after return to the intensive care unit. Arterial and mixed venous blood samples were taken at approximately 30-minute intervals from the peripheral arterial and pulmonary arterial catheters. Sampling was avoided if a change in ventilatory or hemodynamic support was made within 15 minutes. Blood samples were analyzed for oxygen, carbon dioxide, and lactate levels by using a blood analyzer (Rapidlab 865; Chiron Diagnostics, Halstead, United Kingdom). Cardiac output (CO) was then calculated by using the direct Fick method, according to the following equation:

$$CO = Vo_2/(Cao_2 - Cvo_2),$$

where Cao_2 and Cvo_2 indicate arterial and mixed venous oxygen contents, respectively.

Do_2 and the oxygen extraction ratio (ERo_2) were then calculated by using the following equations:

$$Do_2 = CO \times Cao_2$$

and

$$ERo_2 = Vo_2/Do_2.$$

Statistical analysis. Data are expressed as means ± SD. Comparisons were carried out by using the unpaired 2-tailed *t* test. Correlation between 2 data sets was assessed by using the correlation coefficient. The change of the data over the study period was analyzed with single-factor analysis of variance.

Results

Baseline measures. At entry into the study, central and peripheral temperatures were 36.6°C ± 1.3°C (mean ± SD) and 31.8°C ± 2.9°C, respectively. Vo_2 was 142.8 ± 30.4 mL · min^{-1} · m^{-2}, cardiac output was 2.6 ± 0.8 L· min^{-1} · m^{-2}, and Do_2 was 443.0 ± 113.6 mL · min^{-1} · m^{-2}; therefore the systemic oxygen extraction ratio was 0.33 ± 0.07. Although the initial Vo_2 was related to central temperature (r = 0.65), it showed weak correlation with either the duration of cardiopulmonary bypass (r = 0.21) or aortic crossclamping (r = 0.18), the depth of hypothermia (r = 0.11), the rewarming time on bypass (r = 0.15), or the temperature at the end of bypass (r = 0.14).

The arterial blood lactate level measured on arrival was 1.8 ± 0.8 mmol/L, with levels of less than 2.0 mmol/L in 11 patients, 2.0 to 3.0 mmol/L in 8 patients, and 3.8 mmol/L in 1 patient. There were no significant differences between the patients with blood lactate levels of less than 2 mmol/L and those with blood lactate levels of 2 to 3 mmol/L with respect to age, baseline Vo_2, Do_2, central temperature (Table II), or the interval between the time of termination of bypass and the time of sampling (109.4 ± 27.6 minutes for the group with normal lactate levels and 104.3 ± 17.6 minutes for the group with high lactate levels). However, patients with initial lactate levels of less than 2 mmol/L tended to have had shorter cardiopulmonary bypass and aortic crossclamp times and a deeper level of hypothermia during the operation (Table III), although differences did not achieve statistical significance.

Change during the study period. For the group as a whole, Vo_2 increased during the study period to 163.8 ± 30.5 mL · min^{-1} · m^{-2} (P = .001, Fig 1), achieving peak levels between the first and second hour of the study in 6 patients and between the second and fourth

Table II. *Oxygen transport data in patients with lactate levels of less than 2 mmol/L and between 2 and 3 mmol/L on arrival and at 2 hours in the intensive care unit (19 patients)*

	On arrival			*At 2 hours*		
	Lactate <2 mmol/L (n = 11)	*Lactate >2 mmol/L (n = 8)*	P *value*	*Lactate <2 mmol/L (n = 16)*	*Lactate >2 mmol/L (n = 3)*	P *value*
Age (y)	3.7 ± 1.95	3.7 ± 3.4	.99	4.0 ± 2.6	2.0 ± 1.8	.17
Central temperature (°C)	36.7 ± 0.9	36.5 ± 1.9	.80	37.9 ± 0.7	38.4 ± 0.8	.40
V_{O_2} (mL · min^{-1} · m^{-2})	152.1 ± 20.4	134.6 ± 38.9	.27	162.6 ± 26.5	149.3 ± 36.7	.60
D_{O_2} (mL · min^{-1} · m^{-2})	458.6 ± 61.4	446.9 ± 153.1	.84	521.7 ± 113.4	424.1 ± 191.3	.47
Oxygen extraction	0.34 ± 0.06	0.31 ± 0.07	.48	0.32 ± 0.07	0.38 ± 0.10	.43

Values are means ± SD.

Table III. *Cardiopulmonary bypass data in patients with lactate levels of less than 2 mmol/L and greater than 2 mmol/L on arrival in the intensive care unit*

	Lactate <2 mmol/L (n = 11)	*Lactate >2 mmol/L (n = 9)*	P *value*
Cardiopulmonary bypass time (min)	58.1 ± 20.5	86.9 ± 43.9	.10
Aortic crossclamp time (min)	41.3 ± 19.2	54.1 ± 29.4	.28
Rewarming time on cardiopulmonary bypass (min)	23.7 ± 5.5	31.9 ± 17.2	.20
Lowest temperature (°C)	28.5 ± 2.9	25.4 ± 4.2	.19
Temperature at end of cardiopulmonary bypass (°C)	36.8 ± 0.5	36.9 ± 0.5	.61

Values are means ± SD.

hour in the remaining 14 patients. The maximum increase in V_{O_2} during the study period showed weak correlation with cardiopulmonary bypass time, aortic crossclamp time, the depth of hypothermia, rewarming time on bypass, or temperature at the end of bypass.

Both central and peripheral temperature also increased to 38.4°C ± 0.7°C and 35.9°C ± 2.2°C, respectively (both $P = .001$, Fig 1). Comparing between patients, the peak increase in V_{O_2} during the study period was significantly correlated with the peak increase in central temperature ($r = 0.73$, $P = .001$) but not with the peak increase in peripheral temperature ($r = 0.34$, $P = .14$; Fig 2). There was also a close temporal relationship between the peak of V_{O_2} and the highest recorded central temperature. As a result, for every increase in central temperature of 1°C, V_{O_2} increased by 11.3% ± 8.0%.

Cardiac output increased to 3.4 ± 1.1 mL · min^{-1} · m^{-2} ($P = .001$), and D_{O_2} increased to 546.1 ± 166.2 mL · min^{-1} · m^{-2} ($P = .001$, Fig 1). Oxygen extraction was more variable but tended to decrease slightly over time ($P = .1$, Fig 1). The peak increase in V_{O_2} was significantly correlated with the peak increase in D_{O_2} ($r = 0.64$, $P = .002$). There was a variable change in oxygen extraction at the time of peak V_{O_2} compared with the baseline values ($r = 0.001$, $P = 1.0$; Fig 2).

Overall, the mean arterial blood lactate level decreased to 1.5 ± 0.7 mmol/L ($P = .001$, Fig 1). Among those with lactate levels of 2.0 to 3.0 mmol/L on arrival, 3 had persistently high levels (2-3 mmol/L) during the first 2 hours, but in each it had fallen below 2 mmol/L by the end of the study period. In those with elevated lactate levels at 2 hours, V_{O_2}, D_{O_2}, and oxygen extraction levels in this subgroup at this time were similar to those with lactate levels of less than 2 mmol/L (Table II).

The patient with the highest lactate level at the beginning of the study had a peak level of 6.5 mmol/L at 90 minutes and was the only patient with a significant elevated level at the end of the study (3.9 mmol/L). In this 15-year-old patient, V_{O_2} increased progressively by 25.5% from 106.1 mL · min^{-1} · m^{-2} on arrival. D_{O_2} was initially low (241.3 mL · min^{-1} · m^{-2}), and there was a large central-to-skin temperature difference (9.6°C). During the study, D_{O_2} increased by 91.7%, and oxygen extraction decreased by 34.1% (from 0.44 to 0.29). Although she required substantial inotropic and vasodilator support in the early postoperative period, she was successfully extubated at 10 hours after arrival in the intensive care unit.

In the 11 patients with normal lactate levels, inotropic support was used in 8 patients, with dopamine (3-6 μg · kg^{-1} · min^{-1}) in 7 patients and with dobutamine (3 μg · kg^{-1} · min^{-1}) in the other. In the 9 patients with a lactate level of more than 2.0 mmol/L, dopamine (2-10 μg · kg^{-1} · min^{-1}) was used in 4 patients, and dobutamine (6 μg · kg^{-1} · min^{-1}) was used in 2.

Age and Vo_2. To correct for the potential confounding effects of temperature, the Vo_2 measured when the core temperature was closest to 37°C (36.5°C-38.0°C; any time during the 4-hour period) was taken to analyze its relationship with age (Fig 3). In patients older than 3 months, a close negative correlation was found between age and Vo_2 (r = –0.76). However, the 2 patients aged less than 3 months had a significantly lower Vo_2 (104.4 and 107.1 mL · min^{-1} · m^{-2}, respectively) compared with the rest of the group.

Discussion

This is the first study to investigate Vo_2, as well as its relationship with Do_2, oxygen extraction, and blood lactate levels during the early hours after cardiac operations in children. Our data demonstrate that Vo_2 increases in the early postoperative period and that this increase appears to be closely related to changes in central temperature. Second, increases in Vo_2 are closely paralleled by changes in Do_2, and therefore the oxygen extraction varies little during this period. Third, mild elevations in serum lactate, which appear to be common, do not reflect a deranged Vo_2 /Do_2 relationship and do not predict a difficult postoperative course.

Our study confirms that there is a wide variability of Vo_2 in children after cardiac operations, although over all our average values of Vo_2 were lower than those previously reported in sedated children in the early postoperative period.[4] This may be related to the effects of paralysis in our children, all of whom were heavily sedated and paralyzed with a vecuronium infusion during the study to obviate the confounding effects of movement, agitation, and pain on Vo_2 and to allow a more precise analysis of its determinants.

The determinants of Vo_2

Age. It is widely known that Vo_2 expressed as a function of body surface area is greater in younger than in older awake children.[6] This relationship has also been reported in sedated children with congenital heart malformations during cardiac catheterization[14] and in anesthetized children before a cardiac operation.[4] This relationship seems to be less obvious in studies in children younger than 3 months[14] and some infants,[4] in whom Vo_2 may be significantly lower than expected. A similar trend was seen in our patients after cardiac bypass operations, with an inverse relationship between age and Vo_2 for those over 3 months when measured at normothermia.

Body temperature and cardiopulmonary bypass with hypothermia. In early experimental studies performed with adult volunteers, an increase of 1°C in body temperature was shown to increase Vo_2 by 13%.[15] A relationship between body temperature and the increase in Vo_2 has also been demonstrated in adults and children after cardiac bypass operations.[1,4] Our data reinforces these previous observations, with an approximate 11%

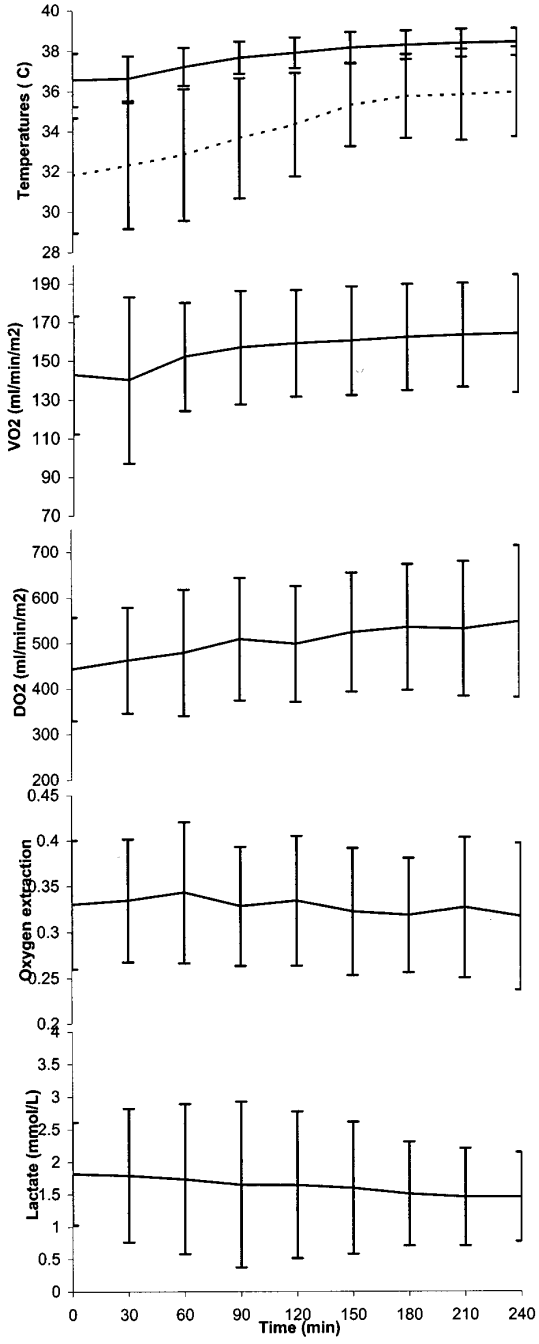

Fig 1. Mean ± SD values of central and peripheral temperatures, oxygen consumption, oxygen delivery, oxygen extraction, and arterial blood lactate levels during the first 4 hours after admission to the pediatric intensive care unit after cardiac bypass operations in 20 children. In the panel concerning temperatures, the *solid line* indicates central temperature, and the *dotted line* indicates peripheral temperature.

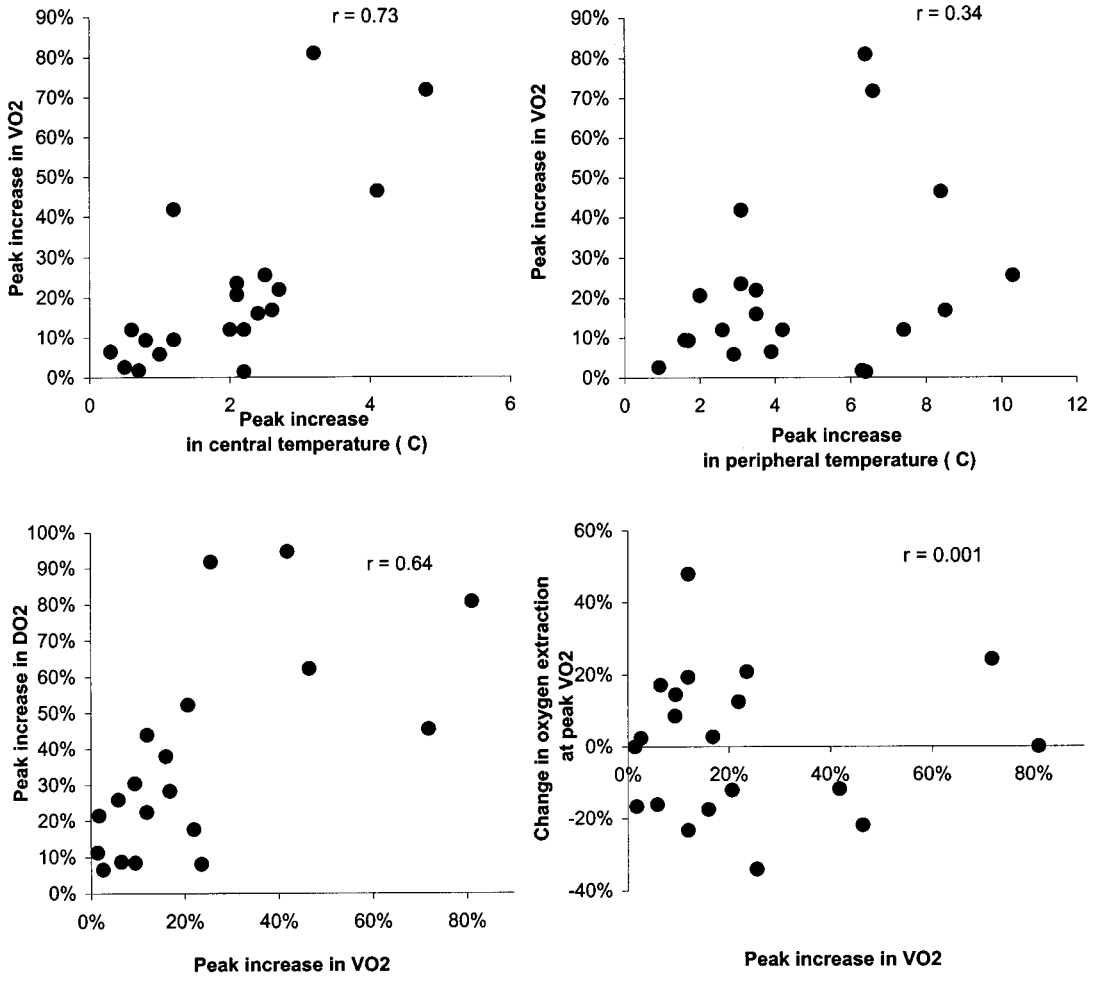

Fig 2. The relationships between peak increase in VO_2 and peak increase in central and peripheral temperatures *(top),* between peak increase in VO_2 and peak increase in DO_2 *(bottom, left),* and between peak increase in VO_2 and the change in oxygen extraction at the time of peak VO_2 *(bottom, right)* during the first 4 hours after admission to the pediatric intensive care unit after cardiac bypass operation in 20 children.

rise in VO_2 per degree of central temperature. Furthermore, there was a close temporal relationship between the peak of VO_2 and the highest recorded central temperature. Because no external heat supply was used in all but 2 patients, our results demonstrate the relationship of VO_2 and central body temperature under natural rewarming conditions after hypothermic cardiopulmonary bypass.

The increase in central body temperature may be attributed to 2 factors: normal homeostatic rewarming and fever induced by the inflammatory response after hypothermic cardiopulmonary bypass. Although central temperature is returned to normal by active rewarming at the end of cardiopulmonary bypass, muscle and subcutaneous fat remain hypothermic, and therefore only 65% of the heat loss is returned to the patient by active rewarming.[16] In our patients central body temperature was 36.8°C at termination of bypass and was 36.6°C (mean) on arrival in the intensive care unit. Subsequently, the time taken to reach a central body temperature of 38°C varied from 1 to 4 hours. The increase in VO_2 during this time presumably reflects the need to generate heat and the catabolic effects of fever and inflammation. Attempts to reduce this increase in VO_2 during rewarming from hypothermic cardiopulmonary bypass have been made. Extended active rewarming during bypass failed to reduce the increase in VO_2 postoperatively in one study,[17] but in other studies provision of an external heat supply was reported to reduce the oxygen uptake

in adults during the rewarming period.[18,19] Our study also showed no correlation between active rewarming time or the temperature at the end of bypass with the increase of V_{O_2}, and because only 2 patients were treated with an external heat supply, no comment regarding the use of external heating in children can be made.

A postoperative fever or temperature overshoot (>38°C) was seen in 15 of 20 children. Fever is one of the signs of the systemic inflammatory response induced by cardiopulmonary bypass.[20,21] It has been reported that this inflammatory response may help to explain the 50% variability of V_{O_2}.[2] It follows, but remains to be demonstrated, that strategies that reduce systemic inflammatory response[22] should reduce the increase in V_{O_2}. A more pragmatic approach may be to actively reduce the central temperature. Moat and associates[23] induced moderate hypothermia (32°C-33°C) in children with refractory low cardiac output states after a cardiac operation to reduce systemic, as well as myocardial, oxygen consumption. V_{O_2} was not measured in this study, but end-organ function appeared to improve with this strategy. Clearly, however, a more formal assessment of the relationship between cardiac output, V_{O_2}, and central temperature is required before this can be recommended as routine therapy.

Relationship between V_{O_2}, D_{O_2}, and oxygen extraction. The increase in V_{O_2} was accompanied with an increase in D_{O_2}, although with variable changes in oxygen extraction. This finding is in agreement with that of one study[9] but different from findings in some other studies,[1,4,10] which found that the increase in V_{O_2} depends mostly on the capacity of tissue oxygen extraction, whereas the relation with cardiac output is relatively weak because of impaired cardiac function after a cardiac operation. This may be because our group of children had a relatively stable cardiac function preoperatively. However, the close relationship between V_{O_2} and D_{O_2} as such should not be interpreted as the so-called V_{O_2} and D_{O_2} dependency phenomenon. To demonstrate this phenomenon in individual patients would first require independent measurement of V_{O_2} and D_{O_2} and would secondly require repeated determinations of V_{O_2} during imposed acute changes in D_{O_2} over a limited period of time. These were not done in the present study.

Arterial blood lactate and oxygen balance. Blood lactate levels are commonly measured to assess the adequacy of tissue perfusion and the balance between oxygen supply and demand in patients after cardiac bypass operations. Mild hyperlactatemia is relatively common after cardiopulmonary bypass, typically with a range of 2 to 4 mmol/L,[3,9-11] although higher levels (>5.0 mmol/L)

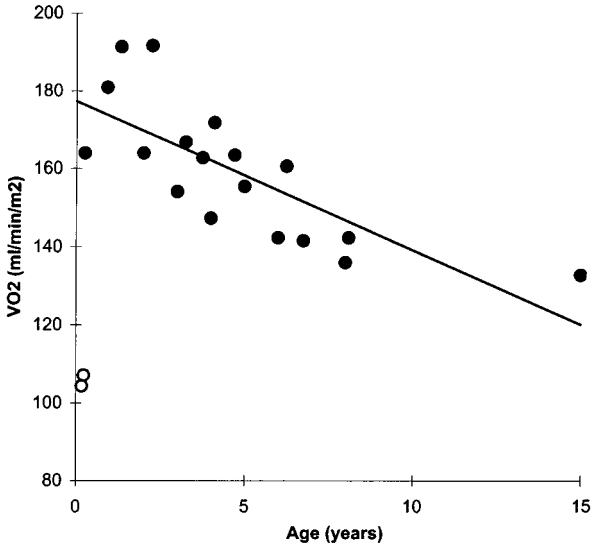

Fig 3. The relationship between oxygen consumption and age when central temperature was closest to 37°C during the first 4 hours after admission to the pediatric intensive care unit after cardiac bypass operations in 20 children. In the 18 patients older than 3 months *(filled circles)*, it shows a trend with a *y* value of –3.8155x + 177.34 and an *r* value of –0.756. The other 2 patients younger than 3 months *(open circles)* had a significantly lower V_{O_2}.

have been reported.[24-26] Only one of our patients fell into this category. This patient had a low D_{O_2} (241.3 mL · min^{-1} · m^{-2}), a large central-to-skin temperature gradient, and clinical evidence of a low cardiac output early after an operation. The lactate level fell as the D_{O_2} increased in response to inotropic and vasodilator support and surface rewarming. It is also likely that this lactate acidosis was associated with a defect of tissue oxygen extraction because despite a 91.7% increase in D_{O_2}, V_{O_2} increased by only 25.5%, and the lactate level remained significantly elevated at the end of the study period. The lactate acidosis in this case should not be confused with the so-called type B lactate acidosis[24] described in other postoperative studies.

The pathophysiology of the more modest hyperlactatemia seen frequently after cardiac operations remains uncertain. A relationship between plasma lactate level and both cardiac output[27] and outcome[25,26] has been suggested, but at the levels measured in our studies, this was not the case. There was a tendency for the duration of cardiopulmonary bypass, aortic cross-clamp time, and the depth of intraoperative hypothermia to be greater in the patients with lactate levels of greater than 2.0 mmol/L on arrival, which is in keeping with the findings of others,[24] but the difference was sta-

tistically insignificant in our study. If absolute cardiac index is not important, then its distribution may be a factor. The gastrointestinal tract is suggested to be the major source of lactate around the time of cardiopulmonary bypass,[28] and splanchnic ischemia has been found to persist during the first few postoperative hours from studies on the basis of gastric tonometry.[29,30] What is clear, however, is that a modestly elevated blood lactate level does not predict more important failure of global DO_2 and cannot be used either as an index of cardiac output or expected outcome.

Limitations of our study. Our study was performed in relatively stable and older children. No neonates with circulatory arrest with hypothermia were included. In addition, because our values for systemic oxygen delivery were derived from the measured oxygen consumption, the potential for mathematical coupling limits our ability to analyze the relationship between these parameters regarding the phenomenon of delivery-dependency of oxygen consumption.

Conclusions

VO_2 is closely correlated with age in children after cardiac operations. The increase in VO_2 during the first 4 hours after return to the intensive care unit is mainly attributed to the increase of central body temperature. Mild elevations in blood lactate levels are common and do not appear to reflect cardiac output, DO_2 or VO_2, or a more complicated postoperative recovery.

We thank Dr Michael Rigby and Dr Elliott Shinebourne for allowing us to study their patients. The assistance of the clinical team is greatly appreciated. We also thank the team from the Biomedical Engineering Department and Ray Matcham of Pfeiffer Vacuum UK for their technical support. Finally, we also thank Dr Simon Crick for helpful discussion and his criticism of this manuscript.

REFERENCES

1. Chiara O, Giomarelli PP, Biagioli B, Rosi R, Gattinoni L. Hypermetabolic response after hypothermic cardiopulmonary bypass. Crit Care Med 1987;15:995-1000.
2. Oudemans-van Straaten, Jansen PGM, Beenakkers ICM, Stoutenbeek CP, van Deventer SJG, Sturk A, et al. Increased oxygen consumption after cardiac surgery is associated with the inflammatory response to endotoxemia. Intensive Care Med 1996;22:294-300.
3. Fiaccadori E, Vezzani A, Coffrini E, Guariglia A, Ronda N, Tortorella G, et al. Cell metabolism in patients undergoing major valvular heart surgery: relationship with intra and postoperative hemodynamics, oxygen transport, and oxygen utilization patterns. Crit Care Med 1989;17:1286-92.
4. Puhakka K, Rasanen J, Leijiala M, Peltola K. Oxygen consumption following pediatric cardiac surgery. J Cardiothorac Vasc Anesth 1994;8:642-8.
5. Chang AC, Kulik TJ, Hickey PR, Wessel DL. Real-time gas-exchange measurement of oxygen consumption in neonates and infants after cardiac surgery. Crit Care Med 1993;21:1369-75.
6. Lewis RC, Dural AM, Iliff A. Standards for basal metabolism in normal infants. J Pediatr 1943;23:1-18.
7. Miline EMG, Elliott MJ, Pearson DT, Holden MP. The effect on intermediary metabolism of open-heart surgery with deep hypothermia and circulatory arrest in infants less than 10 kilograms body weight: a preliminary study. Perfusion 1986;1:29-40.
8. Benzing G III, Francis PD, Kaplan S, Helmosworth JA, Sperling MA. Glucose and insulin changes in infants and children undergoing hypothermic open-heart surgery. Am J Cardiol 1983;52: 133-6.
9. Routsi C, Vincent JL, Bakker J, De Backer D, Lejeune P, d'Hollander A, et al. Relation between oxygen consumption and oxygen delivery in patients after cardiac surgery. Anesth Analg 1993;77:1104-10.
10. Damask MC, Weissman C, Askanazi J, Rosenbaum SH, Elwyn D, Hyman AI. Do oxygen consumption and carbon dioxide production affect cardiac output after cardiopulmonary bypass? Arch Surg 1987;122:1026-31.
11. Ariza M, Gothard JW, Macnaughton P, Hooper J, Morgan CJ, Evans TW. Blood lactate and mixed venous-arterial PCO_2 gradient as indices of poor peripheral perfusion following cardiopulmonary bypass surgery. Intensive Care Med 1991;17:320-4.
12. Shekerdemian LS, Shore DF, Lincoln C, Bush A, Redington AN. Negative-pressure ventilation improves cardiac output after right heart surgery. Circulation 1996;94:II49-55.
13. Davies NJH, Denison DM. The measurement of metabolic gas exchange and minute volume by mass spectrometry alone. Respir Physiol 1979;36:261-7.
14. Lundell BPW, Casas ML, Wallgren CG. Oxygen consumption in infants and children during heart catheterization. Pediatr Cardiol 1996;17:207-13.
15. Shapiro H, Stoner EK. Body temperature and oxygen uptake in man. Ann Phys Med 1966;8:250-5.
16. Davis F, Parimelazhagan K, Harris E. Thermal balance during cardiopulmonary bypass with moderate hypothermia in man. Br J Anaesth 1977;49:1127-32.
17. Joachimsson PO, Nystrom SO, Tyden H. Postoperative ventilatory and circulatory effects of extended rewarming during cardiopulmonary bypass. Can J Anaesth 1989;36:9-19.
18. Joachimsson PO, Nyström SO, Tydén H. Postoperative ventilatory and circulatory effects of heating after aortocoronary bypass surgery: postoperative external heat supply. Acta Anaesthesiol Scand 1987;31:532-42.
19. Cavaliere F, Martinelli L, Guarneri S, Varano C, Coricello M, Sciarra M, et al. Haemodynamic and metabolic effects of surface rewarming after coronary revascularization. Eur J Cardiothorac Surg 1995;9:582-6.
20. Westaby S. Organ dysfunction after cardiopulmonary bypass: a systemic inflammatory reaction by the extracorporeal circuit. Intensive Care Med 1987;13:89-95.
21. Knudsen F, Anderson LW. Immunological aspects of cardiopulmonary bypass. J Cardiothorac Vasc Anesth 1990;4:245-58.
22. Butler J, Rocker GM, Westaby S. Inflammatory response to cardiopulmonary bypass. Ann Thorac Surg 1993;55:552-9.
23. Moat NE, Lamb RK, Edwards JC, Manners J, Keeton BR, Monro JL. Induced hypothermia in the management of refractory low

cardiac output states following cardiac surgery in infants and children. Eur J Cardiothorac Surg 1992;6:579-84.

24. Raper RF, Cameron G, Walker D, Bowey CJ. Type B lactic acidosis following cardiopulmonary bypass. Crit Care Med 1997;25:46-51.
25. Duke T, Butt W, South M, Karl TR. Early markers of major adverse events in children after cardiac operations. J Thorac Cardiovasc Surg 1997;114:1042-52.
26. Cheifetz IM, Kern FH, Schulman SR, Greeley WJ, Ungerleider RM, Meliones JN. Serum lactates correlate with mortality after operations for complex congenital heart disease. Ann Thorac Surg 1997;64:735-8.
27. Ariza M, Gothard JW, Macnaughton P, Hooper J, Morgan CJ, Evans TW. Blood lactate and mixed venous-arterial P_{CO_2} gradient as indices of poor peripheral perfusion following cardiopulmonary bypass surgery. Intensive Care Med 1991;17:320-4.
28. Landow L, Phillips DA, Heard SO, Prevost D, Vandersalm TJ, Fink MP. Gastric tonometry and venous oximetry in cardiac surgery patients. Crit Care Med 1991;19:1226-33.
29. Landow L. Splanchnic lactate production in cardiac surgery patients. Crit Care Med 1993;21(Suppl 2):S84-91.
30. Uusaro A, Ruokonen E, Takala J. Splanchnic oxygen transport after cardiac surgery: evidence for inadequate tissue perfusion after stabilization of hemodynamics. Intensive Care Med 1996; 22:26-33.